COMMENTS REGARDING SCIENTIFIC CLAIMS CONTAINED IN THE REPORT OF THE MARYLAND OCCUPATIONAL SAFETY AND HEALTH ADVISORY BOARD REGARDING TOBACCO SMOKE IN THE WORKPLACE

In its review, the Maryland Occupational Safety and Health Advisory Board failed to acknowledge the vast amount of scientific data that are not consistent with their conclusions. Following are rebuttals to the scientific arguments made in the "Report of the Maryland Occupational Safety and Health Advisory Board Regarding Tobacco Smoke in the Workplace."

1. **The Board cites, as its "primary evidence" of an association between ETS exposure and lung cancer in nonsmokers, the report of the United States Environmental Protection Agency titled Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders, which was issued in December 1992.**

General Comments:

The EPA Risk Assessment provides no basis for regulatory action by the Maryland Occupational Safety and Health Administration (MOSH). The risk assessment has received various criticisms from the scientific community. One criticism is that the EPA's Risk Assessment did not rely upon workplace data and instead, the authors based their conclusions upon data on home exposures of nonsmoking women married to smokers. Another problem is that the EPA Risk Assessment contains no original research. The EPA simply reviewed available data on ETS and compiled those data in a meta-analysis. The statistical procedures utilized by the EPA in the risk assessment have also been questioned in the scientific community.

The EPA's overall handling of the risk assessment has been challenged in federal court. The plaintiffs contend in part that the EPA's Risk Assessment on ETS was fundamentally flawed and should be declared null and void by the courts because (i) the EPA lacked the statutory authority to take the action it did, (ii) the EPA did not follow its own procedures, (iii) the EPA's prejudgment of the issues violated fundamental fairness and due process and most importantly, (iv) the EPA's biased handling of the science was arbitrary and capricious.

Specific Comments:

The EPA Risk Assessment employs a Population-Attributable Risk model for estimating excess lung cancer mortality among

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nonsmokers reportedly exposed to ETS. This model is based upon three estimates:

1. a point estimate or relative risk derived from a meta-analysis of epidemiologic studies on nonsmoking wives married to smokers;

2. the proportion of nonsmokers in the general (U.S.) population reportedly exposed to ETS; and

3. the total number of nonsmokers in the general population.

To calculate the Population-Attributable Risk (PAR), the authors of the EPA Risk Assessment estimated that 60% of all nonsmokers are exposed to ETS. The authors performed a meta-analysis of eleven U.S. spousal smoking studies, pooled the results and estimated a relative risk of 1.19. The calculated cumulative risk generates an estimate of 3,000 excess deaths per year among nonsmokers in the U.S.

The PAR method employs estimates of relative risk, population fractions of exposure to ETS and lung cancer death rates for the general nonsmoking population in order to generate an estimate of excess mortality reportedly attributable to ETS exposure. It is important to note at the outset that the PAR model itself does not determine that there is an increased risk of lung cancer among nonsmokers from ETS exposure. Rather, the model assumes a causal relationship between ETS exposure and an increased risk of lung cancer among nonsmokers, based upon increased risks reported in epidemiologic studies on spousal smoking. These reported relative risks are, in turn, assumed to represent true relative risks for the entire population due to ETS exposure.

These critical assumptions have been challenged.[1] The meta-analysis in the Risk Assessment was performed on eleven U.S. spousal smoking studies, none of which originally reported an overall statistically significant risk estimate. Nevertheless, the EPA combined the results of the studies and reported that its risk estimate was statistically significant, obtained by reanalyzing the

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1. Anonymous, "A Brief Summary of the Public Comments Submitted to the U.S. EPA in Response to the External Review Draft EPA/600/6-90/006A," submitted to the U.S. Environmental Protection Agency, Science Advisory Board Meeting, December 4-5, 1990.

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data from the original studies and applying a lower standard for statistical significance (a 90% confidence interval). The EPA justified their choice of the 90% confidence interval as consistent with a "one-tailed" statistical test. The one-tailed test, however, presumes causation: it is designed to show the extent of a purported "effect."

The choice of confidence interval was criticized in a presentation at the October SAB Executive Committee meeting, by James J. Tozzi. In a follow-up letter to the EPA, Tozzi included a meta-analysis of 13 available U.S. studies which is not statistically significant using the standard 95% confidence interval.[2] Tozzi reported a relative risk estimate of 1.07 (95% C.I.: 0.95 to 1.21). The meta-analysis submitted by Tozzi included two additional lung cancer studies that reported overall statistically nonsignificant associations between spousal smoking and lung cancer. The EPA omitted from its risk assessment the data from the NCI-funded Brownson et al., 1992, study, one of the largest and most recent studies on ETS and lung cancer, which reported no increase in risk for spousal smoking.[3] They also omitted the Stockwell et al., 1992, study data.[4] If the EPA had considered the Brownson and Stockwell data, its meta-analysis would not have resulted in a statistically significant increased risk estimate for spousal smoking.

Eleven of the epidemiological studies that the EPA considered also included estimates of workplace exposures, but the EPA did not assess the data from those studies. Ten of those eleven studies reported no statistically significant increased risk for nonsmoking females. If the data on workplace exposures are pooled in a meta-analysis, the risk estimate approximates 1.00 (unity), which indicates no positive association between reported workplace exposures to ETS and lung cancer in nonsmokers.[5]

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2. Tozzi, J.J., letter to Erich Bretthauer, Assistant Administrator for Research and Development, U.S. EPA, December 4, 1992.

3. Brownson, R. C., et al., "Passive Smoking and Lung Cancer in Nonsmoking Women," Am J Public Health 82: 1525-1530, 1992.

4. Stockwell, H. G., et al., "Environmental Tobacco Smoke and Lung Cancer Risk in Nonsmoking Women," JNCI 84: 1417-1422, 1992.

5. Layard, M.W., "Comments on the EPA Review Draft 'Respiratory Health Effects of Passive Smoking: Lung Cancer and Other

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The epidemiologic studies on spousal smoking contain no actual exposure data on ETS. The authors of the Risk Assessment assumed the validity of questionnaire responses about possible exposure to ETS based upon spousal smoking and then generalized those responses to estimate the general population's exposure to ETS. However, a large body of literature exists on actual measurements of ETS in indoor environments, which was only cursorily considered in the Risk Assessment. While these data are independent of the epidemiologic literature, they suggest that nonsmoker exposure to ETS in typical public places and workplaces is minimal.[6] For example, some studies report typical measurements of nicotine ranging from an exposure equivalent of 1/100 to less than 1/1,000 of one filter cigarette per hour.[7]

5.(...continued)
Disorders,'" comments submitted to the EPA Science Advisory Board, June 30, 1992.

6. Sterling, T. and Mueller, B., "Concentrations of Nicotine, RSP, CO and CO_2 in Nonsmoking Areas of Offices Ventilated by Air Recirculated from Smoking Designated Areas," Am Ind Hyg Assoc J 49(9): 423-426, 1988.

Kirk, P., et al., "Environmental Tobacco Smoke in Indoor Air." In: Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 99-112, 1988.

Carson, J. and Erikson, C., "Results from Survey of Environmental Tobacco Smoke in Offices in Ottawa, Ontario," Environ Technol Letters 9: 501-508, 1988.

Sterling, T., et al., "Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments," Journal of Occupational Medicine 26(1): 57-62, 1987.

Hosein, R., "The Relationship Between Pollutant Levels in Homes and Potential Sources." In: Indoor Air Quality in Cold Climates: Hazards and Abatement Measures. D. Walkinshaw (ed.). APCA, Ottawa, Ontario, 250-260, 1985.

7. Oldaker, G. and Conrad, F., "Estimation of the Effect of Environmental Tobacco Smoke on Air Quality Within Passenger Cabins of Commercial Aircraft," Environ Sci Technol 21: 994-999, 1987.

Hinds, W. and First, M., "Concentrations of Nicotine and Tobacco Smoke in Public Places," New England Journal of
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The EPA's proposed classification of environmental tobacco smoke as a "known human carcinogen" was based in part upon the imputed identification and presence in ETS of suspected carcinogens reported in mainstream smoke and/or fresh sidestream smoke. However, the EPA did not review the available published data on either the characterization of, or exposure to, ETS in its hazard identification. ETS is neither chemically nor physically equivalent to either mainstream or sidestream smoke, and it is therefore not scientifically acceptable to treat ETS, mainstream smoke and sidestream smoke as physically and quantitatively similar mixtures.[8]

7.(...continued)

Medicine 292(16): 844-845, 1975.

Badre, R., et al., "Pollution Atmospherique par la Fumee de Tabac (Atmospheric Pollution by Smoking)," Ann Pharm Fr 36(9-10): 443-452, 1978. Translation.

Jenkins, R., et al., "Development and Application of a Thermal Desorption-Based Method for the Determination of Nicotine in Indoor Environments." In: Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 557-566, 1988.

Muramatsu, M., et al., "Estimation of Personal Exposure to Tobacco Smoke with a Newly Developed Nicotine Personal Monitor," Environ Res 35: 218-227, 1984.

Muramatsu, M., et al., "Estimation of Personal Exposure to Ambient Nicotine in Daily Environment," Arch Occup Environ Health 59: 545-550, 1987.

Thompson, C., et al., "A Thermal Desorption Method for the Determination of Nicotine in Indoor Environments," Envir Sci Tech 23: 429-435, 1989.

Foliart, D., et al., "Passive Absorption of Nicotine in Airline Flight Attendants," New England Journal of Medicine 308(18): 1105, 1983.

8. Baker, R. and Proctor, C., "The Origins and Properties of Environmental Tobacco Smoke," Env Int (16): 231-245, 1990.

Rodgman, A., "Environmental Tobacco Smoke," Reg Tox and Pharm 16: 223-244, 1992.

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As discussed above, meta-analysis, a statistical procedure which combines the reported risk estimates from a number of studies to generate an overall estimate of risk, was used in the EPA's Population-Attributable Risk model. The problems and limitations of the use of meta-analysis for epidemiologic studies have been addressed in the scientific literature.[9] For instance, meta-analysis does not account for intrinsic bias and confounding in the individual studies, and it cannot remedy study design flaws. As two German scientists, Heinz Letzel and Karl Uberla, noted: "Combining risk estimates from biased or confounded studies by meta-analysis cannot provide correct answers."

While a docket for written comments was not established for the 1992 Risk Assessment, oral comments were allowed at both the July SAB committee and the October Executive Committee meetings. However, commenters were asked not to reiterate previous points of discussion. One major issue raised by Dr. William J. Butler in July was the EPA's treatment of independent risk factors

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Reasor, M. and Will, J., "Assessing Exposure to Environmental Tobacco Smoke: Is It Valid to Extrapolate from Active Smoke?," Journal of Smoking Related Diseases 2(1): 111-127, 1991.

Guerin, M., et al., The Chemistry of Environmental Tobacco Smoke: Composition and Measurement. Lewis Publishers: Chelsea, Michigan. 1992.

Nystrom, C., et al., "Assessing the Impact of Environmental Tobacco Smoke on Indoor Air Quality: Current Status," Proceedings of the ASHRAE Conference, IAQ '86, Atlanta, Georgia, 213-233, April 20-23, 1986.

9. Fleiss, J.L. and Gross, A.J., "Meta-Analysis in Epidemiology, with Special Reference to Studies of the Association Between Exposure to Environmental Tobacco Smoke and Lung Cancer: A Critique," Journal of Clinical Epidemiology 44(2): 127-139, 1991.

Letzel, H. and Uberla, K., "Meta-Analyses on Passive Smoking and Lung Cancer." In: Indoor Air Quality. H. Kasuga (ed.). Berlin, Heidelberg, Springer-Verlag, 316-322, 1990.

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or potential confounders.[10] The EPA took the position that confounders could not account for the reported association between spousal smoking and nonsmoker lung cancer, since the EPA was unable to find a single confounding factor that could consistently account for the results reported in all of the studies used in the Risk Assessment. Butler criticized the EPA's position and stated that there were many potential confounders that should be considered important, and that it was not necessary (or even reasonable to expect) to identify a single confounder that would apply to all of the individual studies.

Finally, if the EPA had followed its own 1986 guidelines for carcinogen risk assessment, it would have included: (1) a hazard evaluation which would have examined data regarding the physical and chemical characterization of ETS, as well as the results from published animal inhalation studies and human short term tests; (2) an exposure evaluation which would have included and integrated the data from well over 35 studies in the published literature that monitored ETS constituents in the air of public places and workplaces; (3) a dose-response evaluation which would have relied upon the actual data reported in the epidemiologic studies on spousal smoking (and not upon trend tests); and (4) a risk characterization which would have included the range of uncertainty in assumptions and numbers of lung cancer deaths reportedly attributable to ETS exposures. The guidelines also require that chance be ruled out statistically in all epidemiologic studies under consideration and that all possible biases and possible confounding factors are to be considered.

Thus, the conclusions of the EPA's Risk Assessment are based on an incomplete and selective review of the existing data on ETS. Several of the public comments on the 1990 Draft suggested that this amounted to EPA's apparent failure to follow its own guidelines for carcinogen risk assessment.[1] The EPA could not have classified ETS as a Group A carcinogen had it used the methodologies and guidelines it employed in its previous risk assessments.

2. **The Board claims that the EPA's Science Advisory Board was**

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10. Butler, W.J., "Review of: Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders (EPA/600/6-90/006B; SAB Review Draft; May 1992)," comments submitted to SAB Committee meeting, July 23-24, 1992.

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made up of "independent" scientists.

Comments:

Contrary to the Board's claim, virtually all of the members of the EPA Science Advisory Board on ETS have held contracts with the EPA. The chairman of the panel candidly admitted to the press that "[i]t's not that I'm a tool of the industry. I'm a bigger tool of government. I've been working for the EPA longer. I have more to lose by offending EPA than industry."[11] In addition, many have made vocal, public anti-smoking pronouncements. There was not one industry representative on the Science Advisory Board which examined the EPA's risk assessment on ETS. On the other hand, one member of the Science Advisory Board panel included a well-known anti-smoking activist, and other members of the panel have made public pronouncements against smoking and ETS. Still another member of the 1990 SAB panel was part of a research team responsible for one of the largest ETS studies ever conducted. Although the study reported a negative association between the incidence of lung cancer and marriage to a smoker, the SAB member failed to disclose this fact or the results of the study to other SAB members while the Risk Assessment was under consideration.

Moreover, several of the SAB panel members had reviewed or drafted portions of the Risk Assessment and others were responsible for scientific studies that were discussed and cited in the report. Those panelists were thus put in a position of evaluating their own work.

3. **The Board claims that the Science Advisory Board fully considered all issues and critical comments regarding the EPA's risk assessment on ETS.**

Comment:

According to reviewers of the EPA docket, forty percent of the issues raised in the critical comments to the SAB were not even mentioned by the members of the SAB.[12] Members of the SAB publicly acknowledged that they had not reviewed even selected comments to the public docket. Why was there little concordance between the critical reviewers of the Risk Assessment and the SAB

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11. Los Angeles Times, November 24, 1990: A-27

12. Bero, L.A., and Glantz, S.A., "Tobacco Industry Response to a Risk Assessment of Environmental Tobacco Smoke," Tobacco Control 2: 103-113, 1993.

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report? We know that the SAB members did not review the 107 public comments to the EPA docket. The lack of concordance may also indicate the SAB's lack of understanding of the many issues involved.

4. **The Board utilizes the testimony of Donald Shopland and considers him a "highly credible witness."**

Comment:

Donald Shopland's job is the elimination of smoking. The importance of the ETS/health issue to the antismoking movement is demonstrated by the 1986 comment of Shopland, the then-director of the U.S. Office of Smoking and Health, that "of all the issues, this [ETS] is the one that will propel the United States toward a smoke-free society."[13]

5. **The Board considers the 1986 Surgeon General's Report as important "evidence."**

Comment:

The Surgeon General's review has been challenged by a number of critics. One reviewer, Ann Fettner, suggested that the Surgeon General's conclusions were based on "flimsy" evidence presented in an effort to "divert our attention" from important health concerns such as the "poisoning of the environment."[14] A U.S. Congressman, Walter B. Jones, in a letter published in the Congressional Record, wrote that "the conclusions in the Surgeon General's Report are not supported by the research in his own report."[15] For example, the Surgeon General's conclusion that a causal relationship exists between ETS exposure and lung cancer in nonsmokers was based on 13 epidemiologic studies of women whose husbands smoked. Of those studies, eleven reported risk estimates

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13. Shopland, D., Quoted in "The Campaign Against Smoking Gains Momentum" by J.K. Iglehart, New England Journal of Medicine 314(16): 1059-1064, April 17, 1986.

14. Fettner, A., "Where There's Smoke There's Fire -- The New 'Passive Smoking' Rules: Sidestepping Issues by Scapegoating Smokers," Voice, 5-7, March 30, 1987.

15. Jones, W., "Inconclusive Evidence on the Harmful Effects of Smoking," U.S. Congressional Record - Extension of Remarks, E489-E490, February 18, 1987.

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that were not statistically significant overall.[16]

6. **The Board cites the closing paragraph of the Brownson study as providing support for the EPA's conclusions.**

Comment:

While the final paragraph of the Brownson study may provide an opinion regarding smoking in the workplace, the data from the study do not support the EPA's conclusions. The data from the Brownson study indicate no increased risk overall for spousal smoking.

7. **The Board presents and relies upon the arguments and conclusions of A. Judson Wells, Stanton Glantz and Kyle Steenland.**

Comment:

A. Judson Wells, characterized in the Board's report as a former DuPont employee, is a paid consultant to the American Lung Association, an organization dedicated to the elimination of smoking. Wells has well-known antismoking views; however, he is not an authority on tobacco smoke issues.

There have been two published risk assessments of environmental tobacco smoke (ETS) exposure and heart disease, the first by Wells in 1988, and the second by Kyle Steenland in 1992.[17] However, despite his never having published a risk assessment on ETS and heart disease, Stanton Glantz is the individual most often cited for the conclusion that a large number of annual heart disease deaths in the U.S. are attributable to ETS exposure. In a

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16. Schwartz, S.L. and Balter, N.J., "ETS-Lung Cancer Epidemiology: Supportability of Misclassification and Risk Assumptions," Environmental Technology Letters 9: 479-490, 1988.

17. Wells, A.J., "An Estimate of Adult Mortality in the United States from Passive Smoking," Environment International 14(3): 249-265, 1988.

Steenland, K., "Passive Smoking and the Risk of Heart Disease," JAMA 267(1): 94-99, 1992.

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1991 article with William Parmley,[18] Glantz claimed that the number of annual ETS-related nonsmoker deaths in the U.S. is approximately 53,000, the largest portion of which (37,000) he claimed was from heart disease.

The figure of 53,000 annual U.S. deaths attributed to ETS exposure originated in the publication by Wells in 1988, although Wells actually argued for a "best estimate" of ETS related deaths that was somewhat lower, about 46,000, with 32,000 of these due to heart disease. Stanton Glantz and William Parmley repeated the 53,000 figure in their 1991 article in Circulation. However, they did not themselves independently perform a calculation of "excess" deaths, but instead relied on the earlier work by Wells, which is cited by Glantz and Parmley as the basis for their claim.

To calculate excess deaths due to ETS exposure, Wells used meta-analysis to derive ETS-associated relative risks for lung cancer, heart disease and cancers other than the lung. These relative risks, together with an estimate of the fraction of the population exposed to ETS, were entered into an equation that was used to calculate excess death rates for never smokers. To derive a value of actual numbers of deaths related to ETS exposure, these excess death rates were multiplied by the number of people in the U.S. estimated to be exposed. Based on such calculations, Wells estimated that ETS exposure produced 39,000 excess deaths per year in the U.S.

Further calculations attempted to take into account misclassification, either of smokers as nonsmokers or of nonexposed nonsmokers as exposed to ETS. Wells calculated that these adjustments for misclassification resulted in a revision upward of the estimate, from 39,000 to 53,000. Wells then stated that a more "conservative" estimate would be 46,000, a value chosen because it is halfway between the unadjusted value of 39,000 and the adjusted value of 53,000.

It is clear that the claim of 53,000 excess deaths associated with ETS exposure is assailable at every level of its derivation. At the most fundamental level, the studies contributing to the meta-analysis are scientifically flawed. The flaws stem from, among other reasons, small sample sizes, inadequate control for potential confounding factors and unreliable estimates of ETS exposure. Such flaws contribute to the invalidity of the subsequent meta-analysis, a technique which is further

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18. Glantz, S.A. and Parmley, W.W., "Passive Smoking and Heart Disease: Epidemiology, Physiology, and Biochemistry," Circulation 83(1): 1-12, 1991.

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called into question because it is not designed to bring together such methodologically disparate studies. Furthermore, any overall estimate of the prevalence and levels of ETS exposure within the U.S. is based on assumptions, rather than direct measurement. In addition, a variety of other unproven assumptions are made in attempting to adjust the data to take potential misclassification into account. Finally, the seemingly capricious derivation of the annual death rate claim is reflected in Wells' final calculation in which he decided, based on no particular formula, that the value of 46,000 would be a "best estimate," simply because it was halfway between an unadjusted and adjusted estimate. Such loose and unscientific conclusions should not form a basis for regulatory action.

Kyle Steenland, a National Institute for Occupational Safety and Health employee, also performed a risk assessment of ETS and heart disease, published in 1992. He calculated that 35,000-40,000 annual U.S. heart disease deaths are attributable to ETS exposure. He concluded that "heart disease mortality is contributing the bulk of the public health burden imposed by passive smoking."

There are two important differences between Steenland's estimation process and that used by Wells. First, Steenland did not do a meta-analysis to obtain a pooled estimate of relative risk for heart disease mortality associated with ETS exposure. Instead, he simply adopted the relative risk reported in a single study of a Maryland sample and applied that to the entire U.S. population. Second, he focused only on heart disease and did not attempt to calculate ETS-related deaths from other diseases.

Other than the above, Steenland's procedure for calculating deaths attributable to ETS exposure was generally similar to that reported by Wells. This estimation process involved: positing an overall increase in relative risk of heart disease associated with ETS exposure; making adjustments for potential misclassification and for background exposure; estimating the extent of exposure to ETS; and estimating the fraction of nonsmoker heart disease deaths attributable to ETS exposure. These estimates were incorporated into a formula using data on U.S. heart disease death rates and population estimates, from which was derived an estimated number of annual heart disease deaths attributed to ETS exposure. According to Steenland's calculations, "the overall estimate of ETS-attributable heart disease deaths for never-smokers and former smokers is 35000 to 40000." He further commented that these increased risks of death "are higher than those accepted in regulating environmental toxins."

Steenland's calculations involved problems similar to

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those that plagued Wells' attempt to derive a number of deaths associated with ETS exposure. Although Steenland cannot be criticized for performing an invalid meta-analysis (because he did no meta-analysis at all), his procedure can perhaps be criticized even more severely because he took a relative risk based on a single study from a single county in a single state and applied it to the United States as a whole. Criticisms relating to unverified assumptions, which were noted in regard to Wells' calculations, also apply to Steenland's procedure.

8. **The Board downgrades the use of "reviews" in the industry submissions.**

Comment:

This is ironic, especially because the papers relied upon most heavily by the Board in the areas of lung cancer and heart disease (i.e, those by the U.S.E.P.A., Kyle Steenland, Stanton Glantz and A. Judson Wells) are reviews.

9. **The Board appears to suggest that data can be relied upon or used to support a position only if they are "peer-reviewed."**

Comment:

While important, the peer-review process is not infallible and it does not guarantee either the veracity or the integrity of research. It is also a process which is not itself devoid of potential bias or subjectivity. Any scientist who has attempted to publish a study outside of "mainstream" thinking or which reports a negative association is likely to have experienced a peer-review "bias." This is particularly true with regard to papers on smoking and ETS. Moreover, letters to the editor, symposia reports and other examples of non peer-reviewed literature contain valuable information and they frequently provide authors with an alternative forum to present research, new data and interpretation. Scientific truth deals with content and data and not with the vehicle of presentation.

In addition, the Board appears to ignore the fact that the critical comments submitted to it include many peer-reviewed articles. If the Board exercised appropriate scrutiny in reviewing the articles submitted, they would realize that a great deal of the literature submitted is, in fact, peer-reviewed.

10. **The Board cites "evidence" presented by Dr. Rebecca Bascomb in**

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support of the claim that ETS exposure is associated with respiratory and allergic responses in nonsmoking adults.

Comment:

Only two of the seven available clinical studies report objective decreases in lung function among the majority of asthmatics exposed to environmental tobacco smoke. However, the patients in both positive studies were exposed to excessively high and unrealistic levels of ETS, and the authors themselves noted that the observed effects may have been due to psychological factors.[19] In the remaining five clinical studies, a number of patients reportedly complained of subjective symptoms upon exposure to ETS, but objective results, i.e., evidence of airways obstruction or significant changes in lung flow rates, were not observed in the vast majority of patients.[20] Data from the three

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19. Dahms, T., et al., "Passive Smoking Effects on Bronchial Asthma," Chest 80(5): 530-534, 1981.

    Danuser, B., et al., "Effects of a Bronchoprovocation Challenge Test with Cigarette Sidestream Smoke on Sensitive and Healthy Adults," Chest 103(2): 353-358, 1993.

20. Shephard, R., et al., "'Passive' Exposure of Asthmatic Subjects to Cigarette Smoke," Environmental Research 20: 392-402, 1979.

    Ing, A., and Breslin, A., "The Effect of Passive Cigarette Smoking on Asthmatic Patients," Proceedings, The Thoracic Society of Australia, 543, October 1983. Abstract.

    Knight, A., and Breslin, A., "Passive Cigarette Smoking and Patients with Asthma," The Medical Journal of Australia 142: 194-195, 1985.

    Wiedemann, H., et al., "Acute Effects of Passive Smoking on Lung Function and Airway Reactivity in Asthmatic Subjects," Chest 89(2): 180-184, 1986.

    Stankus, R., et al., "Cigarette Smoke-Sensitive Asthma: Challenge Studies," Journal of Allergy and Clinical Immunology 82: 331-338, 1988.

    Magnussen, H., et al., "Effect of One Hour of Passive Cigarette Smoking on Lung Function and Airway Responsiveness in Adults and Children with Asthma," Clin Investig 70: 368-

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population surveys on adult asthmatics are generally consistent with results reported in the majority of clinical studies.[21]

Although some individuals claim to be annoyed by the sight and smell of tobacco smoke and a few even report experiencing irritation, the existence of human allergens in tobacco smoke has not been established scientifically. Claims about tobacco smoke allergy arise because some individuals -- usually those who experience allergic reactions to other substances such as weeds -- develop allergic skin responses from application of tobacco leaf extract or tobacco smoke condensates. However, positive skin reactions to leaf extracts or condensates have not been correlated with clinical responses to tobacco smoke exposures.[22] Indeed,

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371, 1992.

Jorres, R., and Magnussen, H., "Influence of Short-Term Passive Smoking on Symptoms, Lung Mechanics and Airway Responsiveness in Asthmatic Subjects and Healthy Controls," Eur Respir J 5: 936-944, 1992.

21. Lebowitz, M., "The Effects of Environmental Tobacco Smoke Exposure and Gas Stoves on Daily Peak Flow Rates in Asthmatic and Non-Asthmatic Families," ETS-Environmental Tobacco Smoke: Report from a Workshop on Effects and Exposure Levels, eds., R. Rylander, et al. Published simultaneously in European Journal of Respiratory Diseases, Supplement 65(133): 90-97, 1984.

Bailey, W., et al., "Characteristics and Correlates of Asthma in a University Clinic Population," Chest 98(4): 821-828, 1990.

Robbins, A., "Passive Smoking and Chronic Respiratory Disease Symptoms in Non-Smoking Adults," International Journal of Epidemiology 22(5): 809-817, 1993.

22. Stankus, R., et al., "Cigarette Smoke-Sensitive Asthma: Challenge Studies," Journal of Allergy and Clinical Immunology 82: 331-338, 1988.

Stankus, R. and Lehrer, S., "Inhalation Challenge Studies on Tobacco Smoke Sensitive Asthmatics," Indoor and Ambient Air Quality, eds., R. Perry and P. Kirk, London, Selper Ltd., 303-304, 1988.

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studies indicate that people who claim to be "smoke-sensitive" do not react more frequently to tobacco leaf extract than those who do not consider themselves "sensitive" to tobacco smoke.[23]

There are no data suggesting the existence of a specific sensitization of individuals to tobacco smoke.[24] This is not to deny that there are individuals, including those with pre-existent allergies, who may report annoyance and irritation in the presence of ETS. However, the basis for these reactions is neither specific nor immunologic and cannot be regarded as evidence of a genuine tobacco smoke allergy.

11. **The Board does not appear to appropriately acknowledge the differences between sidestream tobacco smoke and environmental**

22.(...continued)
Musmand, J., et al., "Tobacco Smoke Allergy: A Fallacy?" Ann Allergy 70: 55, 1993.

23. Stankus, R., and Lehrer, S., "Inhalation Challenge Studies on Tobacco Smoke Sensitive Asthmatics," Indoor and Ambient Air Quality, eds., R. Perry and P. Kirk, London, Selper Ltd., 303-304, 1988.

Musmand, J., et al., "Tobacco Smoke Allergy: A Fallacy?" Ann Allergy 70: 55, 1993. Abstract.

Lehrer, S., et al., "Immunological Evaluation of Tobacco Smoke 'Sensitive' Individuals by Skin, Radioallergosorbent and Precipitin Tests," J Allerg Clin Immunol 67: 49, 1981.

24. Lehrer, S., et al., "Tobacco Smoke Sensitivity: A Result of Allergy?" Ann Allergy 56: 1-10, 1986.

Bylin, G., "Tobaksallergi -- finns den? (Tobacco Allergy -- Does It Exist?)," Lakartidningen 77(16): 1530-1532, 1980. Translation.

Witorsch, P., "Does Environmental Tobacco Smoke (ETS) Cause Adverse Health Effects in Susceptible Individuals? A Critical Review of the Scientific Literature: I. Respiratory Disorders, Atopic Allergy and Related Conditions," Environmental Technology 13: 323-340, 1992.

Musmand, J., et al., "The Nasal Response to Environmental Tobacco Smoke," J Allergy Clin Immunology 91(1-2): 205, 1993. Abstract.

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tobacco smoke.

Comment:

ETS is not the same as either mainstream or sidestream smoke. As one researcher in tobacco smoke chemistry has observed, "there are profound physical and quantitative chemical differences" among the three kinds of smoke (MS, SS and ETS). There are differences in physical/chemical properties, particle size (and behaviors) and relative concentrations of constituents. Studies indicate that constituents in ETS are hundreds to thousands of times more dilute than in either SS or MS. Concentrations of ETS constituents in real-life situations are often below the limits of detection and measurement for even the most sensitive air monitors.

It has been argued that sidestream smoke contains greater amounts of certain chemical substances than mainstream smoke and that, therefore, ETS is "more toxic" than mainstream smoke. Comparisons of substances in SS with MS, called "SS/MS ratios," are not directly relevant to the ETS issue. A SS/MS ratio ignores the effect of dilution in the ambient air upon the substance in question. As two tobacco smoke chemists report:

> The important question is not the ratio of sidestream/mainstream but rather what is the concentration of the constituent in the indoor environment and how does it compare to levels from sources other than ETS. Studies based solely on observations of fresh sidestream, or highly and unrealistically concentrated ETS should take into account the possible differences between these smokes and ETS found in real-life situations.[25]

Similarly, the 1986 Report of the Surgeon General notes: ". . . SS characteristics, as measured in a chamber, do not represent those of ETS, as inhaled by the nonsmoker under non-experimental conditions."

12. **The Board sets forth its reasoning for believing that elimination of smoking in the workplace is the appropriate choice. The Board cites, as one major factor, the conclusions of the 1986 Surgeon General's Report regarding the separation of nonsmokers and smokers.**

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25. Baker, R. and Proctor, C., "The Origins and Properties of Environmental Tobacco Smoke," Env Int (16): 231-245, 1990.

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Comment:

The 1986 Surgeon General's Report concludes that smoking bans will not only reduce ETS exposures, but will also "alter smoking behavior and public attitudes about tobacco use" (p. 322). The Report further suggests that "over time, this may contribute to a reduction of smoking in the United States" (p. 322). Thus, the underlying motivation for the use of the ETS/health argument is to attain a "smoke-free society by the year 2000."

The Surgeon General's claim that separation of smokers and nonsmokers does not minimize nonsmoker exposure to ETS is without scientific support. Studies aboard commercial aircraft and in offices indicate, contrary to the Surgeon General's Report, that the simple separation of smokers and nonsmokers effectively minimizes nonsmoker exposure to ETS.[26] One recent study, for

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26. Vaughan, W. and Hammond, K., "Impact of 'Designated Smoking Area' Policy on Nicotine Vapor and Particle Concentrations in a Modern Office Building," J Air Waste Manage Assoc 40: 1012-1017, 1990.

Hedge, A., et al., "Building Ventilation and Smoking Policy Effects on Indoor Air Quality and Employee Comfort and Health." In: Indoor Air '90: Proceedings of the Fifth International Conference on Indoor Air Quality and Climate. Toronto, Canada, Volume 1, 739-744, July 29-August 3, 1990.

Sterling, T. and Mueller, B., "Concentrations of Nicotine, RSP, CO and CO_2 in Nonsmoking Areas of Offices Ventilated by Air Recirculated from Smoking Designated Areas," Am Ind Hyg Assoc J 49(9): 423-426, 1988.

Sterling, T., et al., "Exposure to Environmental Tobacco Smoke in the Non Industrial Workplace Under Different Conditions of Ventilation and Smoking Regulation." In: Present and Future of Indoor Air Quality. C.J. Bieva, et al. (eds.). Elsevier Science Publishers, Amsterdam, 111-118, 1989.

Malmfors, T., et al., "Air Quality in Passenger Cabins of DC-9 and MD-80 Aircraft," Environ Technol Letters 10: 613-628, 1989.

Oldaker, G., et al., "Estimation of Effect of Environmental Tobacco Smoke on Air Quality Within Passenger Cabins of Commercial Aircraft II." In: Indoor Air Quality and Ventilation, F. Lunau and G. Reynolds (eds.). London, Selper

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example, reported that the use of designated smoking areas reduced exposure to ETS by 80 percent. Another study of a smoking-restricted office building reported that ambient nicotine in nonsmoking areas was virtually undetectable, suggesting that ETS had a negligible impact on the nonsmoking areas in the building. In addition, Canadian researchers Sterling, et al., in a series of studies, collected data on levels of ETS constituents in offices with different smoking policies. They reported no significant differences in average ETS constituent levels between nonsmoking offices that received recirculated air from designated smoking areas and nonsmoking offices that did not receive recirculated air. They concluded:

> The results indicate that the provision of a designated, but not separately ventilated smoking area can effectively eliminate or drastically reduce most components of environmental tobacco smoke from nonsmoking offices.

The NIOSH Current Intelligence Bulletin No. 54 also adopts the view that simple separation is not adequate. However, the NIOSH CIB, which reaches conclusions about purported risks associated with exposure to ETS in the workplace, is not a comprehensive, critical review of the available data. Of even greater significance, it does not address available workplace data on exposures or potential health effects.

NIOSH and the EPA advocate either smoking bans or the segregation of smokers into rooms with separate ventilation and/or separate exhaust. However, the creation of a dedicated smoking room with separate ventilation and exhaust in an existing building can be cost-prohibitive and, in some circumstances, physically impossible. The smoking ban is therefore represented by anti-smokers as the most "cost-effective" means of eliminating ETS

26.(...continued)
Ltd., 447-454, 1990.

Oldaker, G. and Conrad, F., "Estimation of the Effect of Environmental Tobacco Smoke on Air Quality Within Passenger Cabins of Commercial Aircraft," Environ Sci Technol 21: 994-999, 1987.

Sterling, T., "ETS Concentrations Under Different Conditions of Ventilation and Smoking Regulation." In: Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 89-98, 1988.

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exposure for the nonsmoker.

Both recommendations -- the smoking ban and separately ventilated smoking rooms -- are radical "solutions" based on the untenable and contradictory position that there should be no exposure whatsoever to ETS or any of its constituents. Ordinary air in public places and workplaces contains many of the same substances identified in ETS, even in the complete absence of smoking. Such substances are produced by many common sources and removal of ETS from the ambient air will not eliminate exposures to them. Moreover, both NIOSH and the EPA establish permissible exposures to such airborne substances, many of which are designated as "suspect carcinogens." The permissible exposure limits for the substances in question are typically far greater than any levels generated by ETS.

13. **The Board suggests that Philip Morris submitted a considerable number of documents claiming to establish that there is no scientific proof ETS increases the risk of heart disease.**

Comment:

The literature submitted by Philip Morris challenges the "biologic plausibility" of the potential relationship between exposure to ETS and heart disease in nonsmokers.

14. **The Board suggests that it does not believe that the "health effects of ETS" should be derived by considering cigarette-equivalents.**

Comment:

Cigarette equivalents are illustrative of exposure and speak to the heart of the "biologic plausibility" argument. For example, typical measurements of nicotine range from an exposure equivalent of 1/100 to 1/1,000 of one filter cigarette per hour.[27]

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27. Carson, J. and Erickson, C., "Results from a Survey of Environmental Tobacco Smoke in Offices in Ottawa, Ontario," Environ Technol Letters 9: 501-508, 1988.

Sterling, T., and Mueller, B., "Concentrations of Nicotine, RSP, CO and CO_2 in Nonsmoking Areas of Offices Ventilated by Air Recirculated from Smoking Designated Areas," Am Ind Hyg Assoc J 49(9): 423-426, 1988.

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This means that a nonsmoker would have to spend from 100 to 1,000 hours or more in an office, restaurant or public place in order to be exposed to the nicotine equivalent of a single cigarette.

A suitable internal dose marker for ETS is not available.[28] However, scientists have estimated that ETS-related

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27.(...continued)

Oldaker, G., et al., "Results From Surveys of Environmental Tobacco Smoke in Offices and Restaurants," Indoor Air Quality, eds., H. Kasuga, Springer-Verlag, Berlin, Heidelberg, 99-104, 1990.

Turner, S., "The Measurement of Environmental Tobacco Smoke in 585 Office Environments," Environment Intl 18: 19-28, 1992.

Hinds, W. and First, M., "Concentrations of Nicotine and Tobacco Smoke in an Air-Conditioned Office Building," New England Journal of Medicine 292(16): 844-845, 1975.

Proctor, C., et al., "Measurements of Environmental Tobacco Smoke in an Air Conditioned Office Building," Environ Tech Letters 10: 1003-1018, 1989.

Muramatsu, M., et al., "Estimation of Personal Exposure to Ambient Nicotine in Daily Environment," Arch Occup Environ Health 59: 545-550, 1987.

Thompson, C., et al., "A Thermal Desorption Method for the Determination of Nicotine in Indoor Environments," Envir Sci Tech 23: 429-435, 1989.

Oldaker, G. and Conrad, F., "Estimation of the Effect of Environmental Tobacco Smoke on Air Quality Within Passenger Cabins of Commercial Aircraft," Envir Sci Tech 21: 994-999, 1987.

Hedge, A., et al., "Effects of Restrictive Smoking Policies on Indoor Air Quality and Sick Building Syndrome: A Study of 27 Air-Conditioned Offices," Indoor Air '93 1: 517-522, 1993.

28. Reasor, R. and Proctor, C., "The Origins and Properties of Environmental Tobacco Smoke," Env Int (16): 231-245, 1990.

Scherer, G., "Uptake of Tobacco Smoke Constituents on Exposure to Environmental Tobacco Smoke (ETS)," Clin Investig 70: 352-

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particle uptake in a typical nonsmoker is approximately two-hundredths of one percent (0.02%) that of the active smoker.[29] Other scientists have estimated that ETS "inhaled doses" (based on particles) are 10,000 to 100,000-fold less than average doses calculated for active smokers.[30] The estimated nonsmoker "dose" is equivalent to actively smoking less than one cigarette over the course of a year.

Another scientist recently calculated estimated "retained doses" for nonsmokers using average exposures to various ETS constituents reported in the published literature.[31] The calculated dose for tobacco smoke particles, expressed in terms of "cigarette equivalents" for purposes of illustration, ranged from less than one-half to about four cigarettes per year for "light" and "heavily-exposed" nonsmokers, respectively.

15. **The Board appears to disregard data submitted which suggest that ETS exposures in public places are minimal. In addition, the Board presents no actual exposure data for Maryland.**

28.(...continued)
367, 1992.

Scherer, G., et al., "Urinary Mutagenicity, HydroxyPhenanthrene, and Tioether Excretion After Exposure to Environmental Tobacco Smoke," Indoor Air Quality, eds., H. Kasuga, Springer-Verlag, Berlin, Heidelberg, 138-146, 1990.

29. Arundel, A., et al., "Never-Smoker Lung Cancer Risks From Exposure to Particulate Tobacco Smoke," Environ Int 13: 409-426, 1987.

Adlkofer, F., "Biological Effects After Exposure to ETS," Indoor Air Quality: Symposium, National Academy of Sciences of Buenos Aires, Buenos Aires, 61-76, 1989.

McAughey, J., et al., "Respiratory Deposition of Environmental Tobacco Smoke," Indoor Air '90: The Fifth International Conference on Indoor Air Quality and Climate, Toronto, Canada, 361-366, July/August, 1990.

30. Gori, G., and Mantel, N., "Mainstream and Environmental Tobacco Smoke," Reg Tox and Pharm 14: 88-105, 1991.

31. Holcomb, L., "Indoor Air Quality and Environmental Tobacco Smoke: Concentration and Exposure," Environ Int 19: 9-40, 1993.

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Comment:

There are well over 100 published studies on ETS measurements in the air of public places, workplaces, restaurants and other locations.[32] The studies typically report measurements of one or more constituents of ETS in the air. The most commonly used "markers" for ETS are carbon monoxide (CO), nicotine and respirable suspended particles (RSP). Other measured constituents include volatile organic compounds (VOCs) such as formaldehyde and benzene, nitrogen oxides (NO_x) and nitrosamines.

Although the published studies on ETS exposures vary greatly in sampling conditions, methods and measurement techniques, the data indicate that, overall, nonsmoker exposure to ETS under normal, everyday conditions is minimal. For example, researchers report that there is little difference in ambient levels of carbon monoxide (CO) in smoking and nonsmoking areas of workplaces and public places and in homes with or without smokers.[33] Other studies indicate that ETS contributes only 20-30% of the total particles in the air of a typical public place in which smoking is

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32. Guerin, M., et al., The Chemistry of Environmental Tobacco Smoke: Composition and Measurement, Lewis Publishers: Chelsea, Michigan, 1992.

Holcomb, L., "Indoor Air Quality and Environmental Tobacco Smoke: Concentration and Exposure," Environment Int 19: 9-40, 1993.

33. Guerin, M., et al., The Chemistry of Environmental Tobacco Smoke: Composition and Measurement, Lewis Publishers: Chelsea, Michigan, 1992.

Holcomb, L., "Indoor Air Quality and Environmental Tobacco Smoke: Concentration and Exposure," Environment Int 19: 9-40, 1993.

Kirk, P., et al., "Environmental Tobacco Smoke in Indoor Air," Indoor and Ambient Air Quality, eds., R. Perry and P. Kirk, London, Selper Ltd., 99-112, 1988.

Cox, B. and Whichelow, M., "Carbon Monoxide Levels in the Breath of Smokers and Nonsmokers: Effect of Domestic Heating Systems," J Epidemiol Comm Health 39: 75-78, 1985.

Girman, J. and Traynor, G., "Indoor Concentrations," JAPCA 33(2): 89, 1983.

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permitted.[34]

16. **The Board cites the suggestion by Stanton Glantz that benzo(a)pyrene is "an important component of ETS."**

Comment:

While it has been suggested that sidestream smoke (and by inference, ETS) contains polycyclic aromatic hydrocarbons (PAH) such as benzo(a)pyrene, in a series of papers, German researchers report no significant differences in urinary PAH by-products among nonsmokers exposed to ETS and those not exposed.[35] Diet, however,

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34. Carson, J. and Erikson, C., "Results from a Survey of Environmental Tobacco Smoke in Offices in Ottawa, Ontario," Environ Technol Letters 9: 501-508, 1988.

Sterling, T., et al., "Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments," Journal of Occupational Medicine 26(1): 57-62, 1987.

Sterling, T. and Mueller, B., "Concentrations of Nicotine, RSP, CO and CO_2 in Nonsmoking Areas of Offices Ventilated by Air Recirculated from Smoking Designated Areas," Am Ind Hyg Assoc J 49(9): 423-426, 1988.

Oldaker, G., et al., "Results From Surveys of Environmental Tobacco Smoke in Offices and Restaurants," Indoor Air Quality, ed., H. Kasuga, Springer-Verlag, Berlin, Heidelberg, 99-104, 1990.

Turner, S., "The Measurement of Environmental Tobacco Smoke in 585 Office Environments," Environment Intl 18: 19-28, 1992.

35. Scherer, G., et al., "Urinary Mutagenicity, HydroxyPhenanthrene, and Thioether Excretion After Exposure to Environmental Tobacco Smoke." In: Indoor Air Quality. H. Kasuga (ed.). Springer-Verlag, Berlin, Heidelberg, 138-146, 1990.

Martin, F., et al., "Urinary Excretion of HydroxyPhenanthrenes After Intake of Polycyclic Aromatic Hydrocarbons," Environ Int (15): 41-47, 1989.

Hoepfner, H., et al., "Hydroxy-Phenanthrenes in the Urine of Non-Smokers and Smokers," Toxicology Letters (35): 67-71, 1987.

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was reported to have a profound influence on PAH by-product formation in all study subjects.

In response to Stanton Glantz's claim which involves the extrapolation from roofers exposed to PAH to ETS-exposed workers, one author writes that Glantz and his co-author do not recognize the differences in the composition of PAH. The author suggests that "from the standpoint of chemical composition of PAH exposures determined by nature of product, PAH exposures of roofers are irrelevant to ETS exposure."[36]

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36. Aviado, D.M., "Complex Mixtures of Tobacco Smoke and the Occupational Environment." In: Patty's Industrial Hygiene and Toxicology, 4th Edition, Volume 2, Part A, G.D. Clayton and F.E. Clayton (eds.). John Wiley & Sons, Inc., Chapter 4, pp. 107-148, 1993.

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